

Can Poverty Drive You Mad? 'Schizophrenia', Socio-Economic Status and the Case for Primary Prevention

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For several decades the dominance of a rather simplistic, reductionist and pessimistic 'medical model' has, especially in relation to 'schizophrenia', relegated poverty and its attendant disadvantages (child neglect and abuse, overcrowding, dysfunctional families, etc.) to the role of mere triggers of a supposed, but unproven, genetic predisposition. For seventy years, however, research has repeatedly demonstrated not only that poverty is a powerful predictor of who develops psychosis, and who is diagnosed 'schizophrenic' (with or without a family history of psychosis), but that poverty is more strongly related to 'schizophrenia' than to other mental health problems. This paper argues that an evidence-based resolution to the longstanding debate between 'social causation' and 'social drift' explanations is that the former perspective explains how poverty is a major cause of psychosis and the latter explains how poverty is involved in its maintenance. Poverty is also a predictor of diagnosis and treatment selection, sometimes regardless of actual symptomatology. Evidence is also presented demonstrating that relative poverty may be an even stronger predictor of mental health problems, including 'schizophrenia', than poverty per se. Psychologists are encouraged to pay more attention to the psycho-social causes of their clients' difficulties, to the role of the pharmaceutical industry in perpetuating a narrow 'medical model' and, most importantly in the long run, to the need for primary prevention programmes.

In his 2004 address to the World Conference on the Promotion of Mental Health and Prevention of Mental and Behavioral Disorders, in Auckland, American psychologist George Albee opened with 'I wrote my first paper emphasizing the necessity of prevention of mental disorders nearly 50 years ago'. After demonstrating in some detail the numerical and economic impossibility of trying to provide mental health services to all those who need them, he went on to say:

The principle source of stress worldwide is poverty. Poverty is at the root of many of the stresses that have been identified as the cause of emotional distress. Poverty has been identified as the 'the cause of the causes' (Joffe,

1988).

– Poverty dampens the human spirit creating despair and hopelessness.

– Poverty underlies multiple problems facing families, infants, children, adolescents, adults, and the elderly.

– Poverty directly affects infant mortality, mental retardation, learning disabilities, and drug and alcohol abuse.

– Poverty is a major factor in homelessness.

– Poverty increases the incidence of racial, ethnic, and religious hatred.

– Poverty increases abuse against

women and children.

– Poverty results in suicide, depression, and severe mental illness.

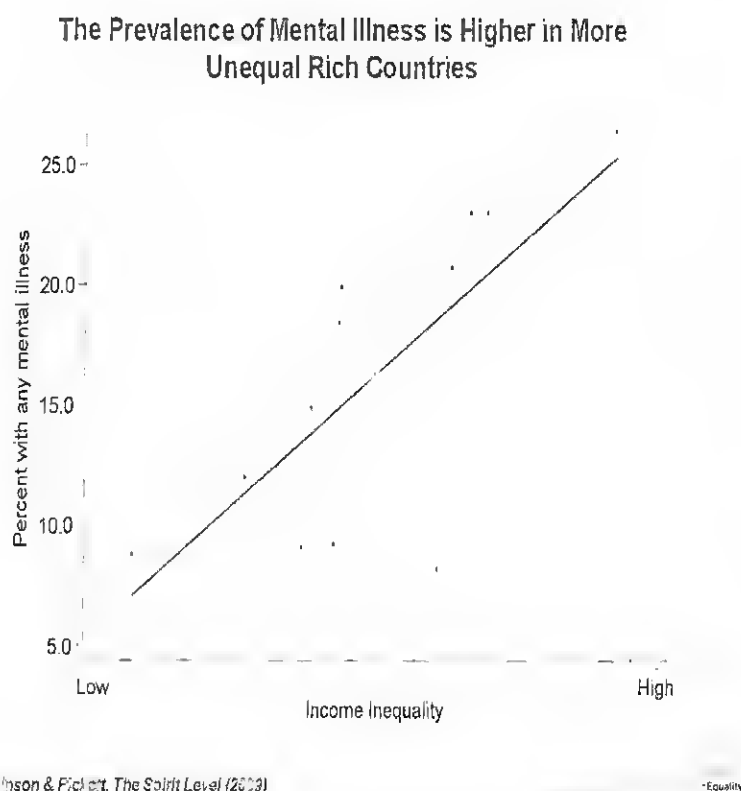
– Poverty is directly linked to violence.

. We believe that the eradication of poverty is the first step in primary prevention. (Albee, 2006, p. 451)

After briefly discussing the relationship between poverty and mental health in general, this paper will (i) examine some of the problems arising from the current dominance of a rather simplistic bio-genetic paradigm, (ii) summarise the extensive research demonstrating the causal relationship between poverty and both psychosis in general and 'schizophrenia' in particular, and (iii) delineate some of the implications at both clinical and primary prevention levels.

The causal role played by poverty in a range of mental health problems is well established, including, for example, in relation to depression (Heflin & Iceland, 2009; Talala, Huurre, Aro, Martelin, & Prattala, 2009), drug abuse (Daniel et al., 2009) and suicidality (Bolton, Belik, Enns, Cox, & Sareen, 2008). The relationship between poverty and poor mental health is, of course, complex and is mediated by variables that are themselves related to poverty such as child abuse and neglect, unemployment, gender, ethnicity, maladaptive coping strategies etc. (Barker-Collo & Read, 2003).

Figure 1



For example, the Christchurch Health and Development study, a prospective birth cohort study of approximately a thousand New Zealanders, found that the elevated rates of mental disorder among Maori involve an interaction between socioeconomic disadvantage and childhood adversity, with secure cultural identity operating as a protective factor (Marie, Fergusson, & Boden, 2008).

Furthermore, poverty (like mental health) can, of course, be measured in multiple ways, at multiple life stages. Another longitudinal birth cohort study, by the Dunedin Multidisciplinary Health and Development Research Unit, found that poverty before age 11 (as well as low IQ at age 5, and antisocial behaviour) predicted PTSD at ages 26 and 32 (Koenen, Moffitt, Poulton, Martin, & Caspi, 2007). The same study also found that while alcohol abuse in adulthood was related to low socioeconomic status in childhood, this was not the case for depression, which was instead related to low socioeconomic status in adulthood (Poulton et al., 2002). A study of over

two million New Zealand adults found that over a three year period the age-adjusted odds ratios for suicide among unemployed people, compared to people in employment, was 2.46 for women and 2.63 for men (Blakely, Collings, & Atkinson, 2003). Another New Zealand study, of over 15,000 families, focussed, rather unusually, on asset wealth (eg home ownership and savings) and found that those in the lowest quintile were three times more likely to report high psychological distress than those in the highest quintile, and that the difference remained statistically significant even after controlling for age, gender, prior health status, and - perhaps surprisingly - income (Carter, Blakely, Collings, Gunasekara, & Richardson, 2009).

It must also be noted that there is very convincing evidence that *relative* poverty is a stronger predictor than poverty per se. In their book, 'The Spirit Level' (and on www.equalitytrust.org.uk), British epidemiologists Richard Wilkinson and Kate Pickett report multiple studies demonstrating a far stronger relationship between relative

poverty and a range of social, health and mental health outcomes than between poverty per se and the same outcomes (Wilkinson & Pickett, 2009). They first note that in many countries rates of mental illness and levels of inequality have both increased significantly in recent decades. They then report a strong relationship between degree of income inequality and rates of people meeting diagnostic criteria for mental illness over a 12 month period, across 11 countries (see Figure 1). Similar findings are reported for other outcomes, including level of use of illegal drugs (Figure 2). In another recent book psychologist and journalist Oliver James coined the term 'Affluenza' to describe and explain this phenomenon (James, 2007).

Problems with Construing Madness as an Illness

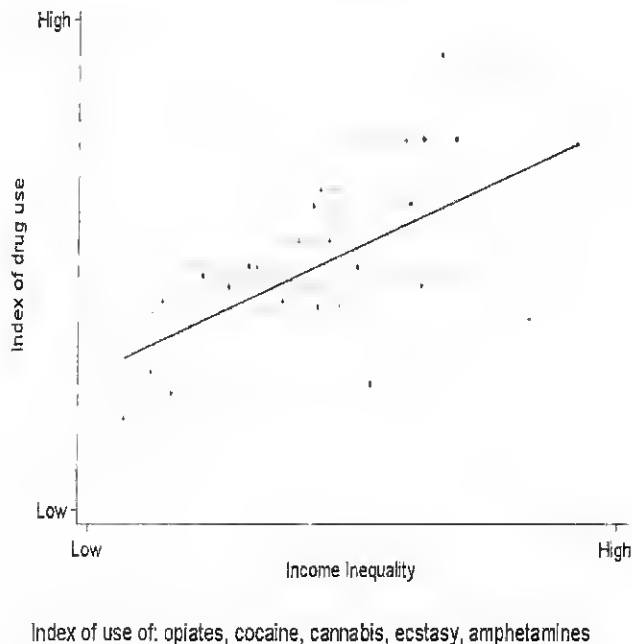
Dominance of the 'Medical Model'

The remainder of this paper will focus primarily on the specific relationship between poverty and 'schizophrenia', a diagnosis traditionally viewed as one of the most severe of 'mental illnesses' and one considered to be particularly bio-genetic in origin and the etiology of which, therefore, is thought to have little to do with social factors such as poverty. I hope to demonstrate that these opinions, which are the cornerstones of the 'medical model' of conceptualising human distress and dysfunction, are not evidence-based.

The paper will argue that the research summarised above, much of which has been undertaken by social scientists, has had remarkably little impact in the field of mental health, and discuss possible reasons for that. As an illustration of how even the most consistent and robust of research literatures can be ignored, minimised or distorted in the service of protecting a dominant paradigm (Kuhn, 1962), particularly one backed by powerful economic interests, we will offer some examples of how the research demonstrating a strong relationship between poverty and 'schizophrenia' (summarised below) has been responded to within some quarters of the mental health community.

Figure 2

Drug Use is More Common in More Unequal Countries

Source: Wilkinson & Pickett, *The Spirit Level* (2009)

Equal, Trust

The dominance, for the past several decades, of a 'medical model' paradigm within the mental health field, especially in relation to the more serious 'illnesses' like 'schizophrenia', has been repeatedly documented and discussed (Bentall, 2003, 2009; Boyle, 1990; Read, Mosher, & Bentall, 2004a; Ross & Pam, 1995). For example, a recent review calculated that while 20.6% of all studies of 'schizophrenia' ever conducted have been concerned with biological causes (and 18.8% with medications) only 0.9% were concerned with poverty and 0.3% with child abuse or neglect. Furthermore the ratio of studies of biological causes to studies of social causes has recently worsened, from 13.3 to one before 2000 to 20.6 to one in the period 2000 to May 2009 (Read, Bentall, & Fosse, 2009). Despite this disproportionate focus on the search for biological causes, there have been no robust findings, in terms of chemical imbalances or other brain abnormalities (Bentall, 2009; Read, 2004a) or, as we shall see below, in terms of a genetic predisposition. PsycINFO searches, in February 2010, reveal that, meanwhile, for many other health and

mental health outcomes, the proportion of studies addressing 'poverty OR socioeconomic status' has *increased* since 2000, including 'major depressive disorder OR depressive disorder' (from 0.8% to 1.6%), 'diabetes' (0.8% to 2.6%) and 'cardiovascular disease OR cardiovascular disorder OR heart disorder' (0.8% to 1.5%).

The Pharmaceutical Industry

Much of the blame for this imbalance has been focussed on psychiatry's inability or unwillingness to resist the increasingly pervasive influence of the pharmaceutical industry, which benefits from promulgating a simplistic, reductionist focus on biological causal factors (Bentall, 2009; Mosher, Gosden, & Beder, 2004; Shooter, 2005). The industry's influence on professional organisations, research funding, drug licensing authorities, psychiatric journals and teaching institutions, which is particularly strong in the U.S.A. (Mosher, et al., 2004), has recently extended to the internet, from which the public (and professionals) increasingly

get their information about the causes of, and treatments for, mental health problems. Recent studies have found that approximately half of all mental health websites are funded by drug companies and that these websites present a more biological perspective about causes and treatments than websites that are free from industry sponsorship, in relation to 'schizophrenia' (Read, 2008), PTSD (Mansell & Read, 2009) and depression (de Wignar & Read, 2009).

In 2005 the then President of the American Psychiatric Association warned:

If we are seen as mere pill pushers and employees of the pharmaceutical industry, our credibility as a profession is compromised. As we address these Big Pharma issues, we must examine the fact that as a profession, we have allowed the bio-psycho-social model to become the bio-bio-bio model (Sharfstein, 2005, p. 3).

The Bio-Psycho-Social Model

Even the 'bio-psycho-social' model, presented in most sources (including psychology and psychiatry textbooks) as a balanced, integrated approach to understanding 'schizophrenia' and other mental health problems, has been distorted by the pressure to conform to the dominant paradigm. The landmark paper introducing the bio-psycho-social model, 'Vulnerability: A new view of schizophrenia' (Zubin & Spring, 1977), had indeed offered the possibility of a genuine integration of psycho-social and biological research. Unfortunately, rather than embrace this opportunity, the momentum of the 'medical model' was so great that the heightened vulnerability to stress, which everyone agreed lay at the core of psychosis, was decreed to be biological in origin (usually genetic but with some attention to perinatal factors). This ignored the fact that Zubin and Spring had clearly stated that there is such a thing as 'acquired vulnerability' and that this can be 'due to the influence of trauma, specific diseases, perinatal complications, family experiences, adolescent peer interactions, and other life events that either enhance or inhibit the development of subsequent disorder'

(p.109).

Since the 1970s the illusion of a balance, of an integration of models, has been created by the so-called 'bio-psycho-social' approach. An integral part of this has been the 'vulnerability-stress' idea that acknowledges a role for social stressors but only in those who already have a supposed genetic predisposition. Life events have been relegated to the role of 'triggers' of an underlying genetic time-bomb. This is not an integration of models, it is a colonisation of the psychological and social by the biological. The colonisation has involved the ignoring, or vilification, of research showing the role of contextual factors such as stress, trauma (inside and beyond the family), poverty, racism, sexism etc. in the etiology of madness (Read, Mosher, & Bentall, 2004b, p. 4).

The attempt to prove the hypothesis underlying biological psychiatry's distorted version of Zubin and Spring's model, i.e. that there is a specific genetic predisposition for something called 'schizophrenia', has probably been one of the most wasteful enterprises ever undertaken by medical researchers. Reviewing the methodologies and concepts deployed in the search for a genetic predisposition shows that there is no robust evidence for it (Joseph, 2006). A recent paper in the *American Journal of Psychiatry* (Sanders et al., 2008), described by the editor as 'The most comprehensive genetic association study of genes previously reported to contribute to the susceptibility to schizophrenia' (S. Hamilton, 2008), found that:

None of the polymorphisms were associated with the schizophrenia phenotype at a reasonable threshold for statistical significance' and that 'of the 69 SNPs (single nucleotide polymorphisms) ... only four showed even nominal association. ... The distribution of test statistics suggests nothing outside of what would be expected by chance (p. 421).

It is testimony to the power of a paradigm, once it establishes dominance, to resist research findings that refute its basic premises (Kuhn, 1962) that these findings are unlikely to prevent the continued consumption of millions of dollars of research funding that might be productively used elsewhere in the mental health field.

The failure to find evidence of a genetic predisposition for psychosis in general, or 'schizophrenia' in particular, can be understood in terms of recently developed knowledge about how epigenetic processes turn gene transcription on and off through mechanisms that are highly influenced by the individual's socio-environmental experiences. To understand the emerging evidence of the relationship between adverse childhood events, including poverty, and subsequent psychosis, it is necessary to integrate these epigenetic processes, especially those involving the stress regulating functions of the HPA axis (Read, Perry, Moskowitz, & Connolly, 2001), with research about the psychological mechanisms by which specific types of childhood trauma can lead to specific types of psychotic experiences (Read, Bentall, Fosse, 2009).

The 'Schizophrenia' Construct

Recent editorials suggest that these 'inconsistent results and disappointing findings of genetic research on 'schizophrenia' arise from 'failure to demonstrate the existence of a unitary disease process' (Ruggeri & Tansella, 2009) and that 'the difficulty in gaining a consistent and clear-cut picture of the genetics of schizophrenia mirrors the marked clinical and neurobiological heterogeneity of the disorder' (Tosato & Lasalvia, 2009).

The construct of 'schizophrenia' is indeed heterogeneous. It is also disjunctive, meaning that one person can receive the diagnosis without having any thing in common with another person with the same diagnosis. It also has little reliability or validity (Bentall, 2003, 2009; Read, 2004b), rendering it very difficult to identify any specific cause, genetic or otherwise. Indeed many researchers are abandoning research into 'schizophrenia', precisely because

of its poor reliability and predictive validity, and are focusing instead on the causes of discrete psychotic phenomena such as hallucinations or delusions (Bentall, 2009). However, much of the research reported below, spanning half a century, predates this recent development and employed the 'schizophrenia' diagnosis.

Summary of the Research

Before summarising the research demonstrating a causal relationship between poverty and 'schizophrenia' (or psychosis or specific psychotic phenomena) it must be noted that, as is the case for the other mental health problems briefly discussed at the outset of this paper, other social factors (many themselves related to poverty) are now known to have a causal role, or to be significant risk factors, for psychosis. These include (often after controlling for family history of 'schizophrenia' or psychosis): mother's health, nutrition and stress during pregnancy; urban birth and urban living; being the product of an unwanted pregnancy; early loss of parents via death or abandonment; separation of parents; witnessing inter-parental violence; dysfunctional parenting (often intergenerational) – particularly affectionless over-control; childhood sexual, physical and emotional abuse; childhood emotional or physical neglect; insecure attachment in childhood; bullying; war trauma; rape or physical assaults as an adult; being a refugee; racist and other forms of discrimination; and heavy marijuana use, especially early in adolescence (Bentall & Femyhough, 2008; Conus, Berk, & Schafer, 2009; Janssen et al., 2003; Janssen et al., 2004; Larkin & Morrison, 2006; Larkin & Read, 2008; Mortensen et al., 1999; Moskowitz, Schafer, & Dorahy, 2008; Parrett & Mason, 2010; Pfeifer et al., 2010; Read, Agar, Argyle, & Aderhold, 2003; Read, et al., 2009; Read & Gumley, 2008; Read, et al., 2001; Read, van Os, Morrison, & Ross, 2005; Schreier et al., 2009; Shevlin, Dorahy, & Adamson, 2007; Shevlin, Murphy, Houston, & Adamson, 2009; Verdoux & Tournier, 2004). The relationship between poverty and 'schizophrenia', therefore,

is 'best understood as being the result of the greater exposure to a range of risk factors, in both childhood and adulthood, which are disproportionately experienced by poorer people.

Mental hospitals and their precursors have always been filled predominantly with poor people (Read, 2004c). In seventeenth century France, for example, the people confined in the Hopital General were almost exclusively the poor of Paris (Foucault, 1965). In the 19th century (prior to the invention of the diagnostic category 'schizophrenia' in 1911 (Read, 2004d) reports had already been emerging that insanity was more common amongst poor people (Bresnahan & Susser, 2003).

The first systematic study, in 1939, found that the deprived central areas of Chicago had higher psychiatric admission rates than the wealthier suburbs (Faris & Dunham, 1939). Contrary to the popular notion that milder mental health problems, but not 'schizophrenia', are socially caused, the difference was particularly high for 'schizophrenia'. People in the poorest areas of Chicago were seven times more likely to be diagnosed 'schizophrenic' than those in the richest parts.

This relationship between poverty and 'schizophrenia' was soon replicated in nine other cities throughout the USA (Clark, 1949). During the 1950s the same relationship was found in Norway, Bristol, Liverpool and London (Kohn, 1976). The famous New Haven study (Hollingshead & Redlich, 1958), which measured class directly on the basis of education and occupation rather than location, found that the poorest class (V: 'unskilled, manual') was three times more likely than the wealthiest two classes (I and II: 'business, professional and managerial') to be treated for psychiatric problems. The diagnosis having the strongest relationship with class was, again, 'schizophrenia'. The poorest people were eight times more likely to be diagnosed 'schizophrenic' than the wealthiest.

By 1976 a review had concluded:

There have been more than 50 studies of the relationship between social class and rates of schizophrenia. Almost without

exception, these studies have shown that schizophrenia occurs most frequently at the lowest social class levels of urban society. The evidence comes from research in Canada, Denmark, Finland, Great Britain, Norway, Sweden, Taiwan, and the United States - an unusually large number of countries and cultures for establishing the generality of any relationship in social science. (Kohn, 1976, p. 177)

Subsequently, a New York study of first admissions found that class V were 12 times more likely to be hospitalised than class I. Furthermore:

The variable related to the most other variables in this population and hence important for understanding many processes involved in the functioning of these patients is membership in class V. None of the clinical variables such as a particular symptom dimension or even level of social functioning relates to as many other variables. . . . This is in striking contrast to the low level of attention often paid to social class in psychiatric practice and research. (Strauss, Kokes, Ritzler, Harder, & VanOrd, 1978, p. 620)

The following year a Tennessee study of 10,000 first admissions again confirmed that the diagnosis most strongly related to low socio-economic status was 'schizophrenia' (Rushing & Ortega, 1979). The relationship between 'schizophrenia' and poverty was described as 'one of the most consistent findings in the field of psychiatric epidemiology' (Eaton, 1980).

More recent research has continued, with some rare exceptions (see Bresnahan & Susser 2003), to confirm the earlier findings, including studies in England (Bristol, Nottingham and London), Wales, Finland, Canada and Nigeria (Read, 2004e). More often than not the strongest relationship between class and psychiatric admission is for 'schizophrenia'. New Zealand studies have produced findings consistent

with the international literature (Abas, Vanderpyl, Robinson, & Crampton, 2003; Kydd, Nola, & Wright, 1991; Salmund & Crampton, 2000).

A British study found that deprived children are four times more likely to develop 'non-schizophrenic psychotic illness' but eight times more likely to grow-up to be 'schizophrenic' than non-deprived children (Harrison, Gunnell, Glazebrook, Page, & Kwiecinski, 2001). Even among children with no family history of psychosis the deprived children were seven times more likely to develop 'schizophrenia', confirming that you do not need a genetic predisposition to develop 'schizophrenia'.

A recent population-based longitudinal study in Israel found that education level of father, education level of mother and occupational status of father (all at birth) were significant risk factors for being diagnosed with 'schizophrenia' (Werner, Malaspina, & Rabinowitz, 2007). However another analysis of the same data found no gradient of risk for 'schizophrenia' associated with social class at birth, and that only the offspring of fathers in the lowest social class had increased risk (Corcoran et al., 2009).

Researchers measuring the more homogenous and reliable constructs of hallucinations and delusions have found, unsurprisingly, that they are more common among people who grew up in poverty (Brown, Susser, Jandorf, & Bromet, 2000).

Some researchers have returned to the methodology of the original Chicago study (Faris & Dunham, 1939) and focused on community (rather than individual) measures of poverty. For example the Israel data reveals that being born in a poor neighborhood is itself a risk factor for 'schizophrenia' (Werner, et al., 2007). Similarly a study of 811 counties across 13 states in the USA found that rates of hospitalization for 'schizophrenia' correlated with percentage of residents living in poverty and percentage unemployed (Fortney, Xu, & Dong, 2009). A New Zealand study found that the relationship between poverty and probability of being admitted to a psychiatric hospital exists even *within* a deprived region. In South Auckland, one of the poorest areas of New Zealand, those living in

the most deprived neighborhoods were, after controlling for age and gender, 4.1 times more likely to be admitted than those living in the least deprived neighborhoods. The most common diagnostic category was 'schizophrenia and schizoaffective disorder' (Abas, et al., 2003). An earlier study, of the whole Auckland region, had found that the highly significant correlation ($p < .0001$) between admission rates and a measure of deprivation (including unemployment, overcrowding, ethnic minority status, and unskilled employment) was largely accounted for by the particularly large correlation in South Auckland (Kydd, et al., 1991).

Psychiatric admission rates increase during economic declines. This relationship is, again, particularly strong for psychoses in general and 'schizophrenia' in particular. (Thompson et al., 2002; Warner, 1985)

These repeated findings, that higher levels of disturbance (of the kind found in supposedly more biologically-based illnesses like 'schizophrenia') are more related to poverty and other social factors than mild or moderate levels of disturbance, is indirectly supported by the New Zealand study referred to in Figure One (Wells et al., 2006). Even without including the psychosis data (which is not reported), this interview survey of nearly 13,000 adult New Zealanders found that those with no education were 1.2 times more likely than those with education beyond school to be diagnosed with any DSM diagnosis, but 1.8 times more likely to be assessed as having a 'serious disorder'; with corresponding rates for the lowest vs highest income groups of 1.7 for 'any disorder' but 2.9 for 'serious disorder'.

Relative poverty

Although psychosis was not included in the studies summarized in Figure One, the authors of *The Spirit Level* point out that 'severe mental illness' is 'strongly correlated with inequality; mood disorders less so' (Wilkinson & Pickett, 2009). There is good evidence to support the relative poverty hypothesis in relation to psychosis. A study of 17 wards in South London found that high inequality (measured by degree of

distribution of a composite deprivation score) was associated with incidence of 'schizophrenia' (after adjusting for age, sex, absolute deprivation, and ethnicity), but only in the group of the most deprived wards (Boydell, van Os, McKenzie, & Murray, 2004). A similar South Africa study, comparing seven municipalities, found no significant relationship between a poverty measure (percentage of residents above or below the national poverty line) and being treated for a first episode of psychosis. However the study did find a significant relationship with inequality, measured by the mean annual income of the top 10% wage earners divided by the mean annual income of the bottom 10% (Burns & Esterhuizen, 2008). The relationship remained significant after controlling for gender, age, ethnicity, urbanicity and employment status.

Explanations

The figures, presented earlier, about the relative dearth of research into poverty and the various disadvantages and adverse life events associated with poverty, indicate that the most common response from the mental health community is not to try and explain the powerful relationship between poverty and madness at all. The most common reaction, it seems, is just to ignore it. Another common response has been to deploy the distorted version of Zubin and Spring's stress-vulnerability model, arguing that if poverty is involved at all in the etiology of 'schizophrenia' then it is only as a trigger or exacerbator of the 'illness' and only in those with the supposed genetic predisposition. Other attempts to explain the relationship follow.

Can madness cause poverty?

One explanation, which protects the dominance of the bio-genetic paradigm, is to argue that 'schizophrenia' afflicts all classes equally but those at the top of the economic pyramid dribble down to the bottom as result of their 'illness'. This is the 'social drift' theory. Because of the paucity of evidence supporting the 'social drift' notion (Read, 2004e), psychiatry has fallen back on weaker variations of the same theme. The

'social selection' theory (Eaton, 1980) suggested that although 'schizophrenics' have not actually drifted downwards themselves, their impoverished circumstances are still a result, rather than a cause, of their 'illness', because they are of a lower social status than their parents, or, weaker still, because they have not climbed up the pyramid as far as they should have – the 'social residue' theory.

Those who argue that the relationship between poverty and 'schizophrenia' is best explained by these theories, rather than by a 'social causation' model, tend not to mention that the New Haven study had tested the 'social drift' theory, by investigating whether 'class V patients had drifted to the slums in the course of their lives' and whether 'schizophrenics' were socially downward mobile. No evidence of such 'social drift' was found. The study also rejected the weaker social selection theory because 91% of the 'schizophrenics' were in the same social class as their parents – rather than a lower class as predicted (Hollingshead & Redlich, 1958). In fact an even earlier 25-year study had already failed to find evidence of social drift and had concluded that 'The excess of psychoses from the poorer area is a product of the life conditions entailed in the lower socio economic strata of the society' (Lapouse, Monk, & Terris, 1956).

A later Canadian study reported measurements of class and psychiatric disorder taken ten years apart and found 'that socio-economic status was more likely to have causal priority over psychiatric disorder than the reverse' (Lee, 1976). This longitudinal approach was repeated in Illinois and Michigan, using multiple points in time. In both states the results 'favour a social causation interpretation' (Wheaton, 1978).

Beyond predicting who becomes 'schizophrenic' in the first place, low 'social class of origin' (at birth or during childhood), which 'cannot be caused by schizophrenia', also predicts negative outcome among people with severe psychosis (Samele et al., 2001).

The relationship between urbanicity and 'schizophrenia' has been shown to be less a consequence of social drift or social residue (Dauncey, Giggs, Baker,

& Harrison, 1993) than a consequence of growing up in the city (Marcelis, Navarro-Mateu, Murray, Seltén, & Van Os, 1998).

Of course there *are* social and economic consequences to being extremely distressed, alienated or disoriented. There are additional consequences, such as stigma – both from without and internalised – which is exacerbated by having one's difficulties explained in terms of having a 'mental illness' (Angermeyer & Matschinger, 2005; Read, 2007; Read, Haslam, Sayce, & Davies, 2006). In addition, anti-psychotic medications, along with any beneficial effects, have a range of adverse effects which can severely limit one's social, cognitive, and occupational functioning ((Bentall, 2009; Ross & Read, 2004; Weinmann & Aderhold, 2010; Weinmann, Read, & Aderhold, 2009). Therefore it would be surprising if the social drift hypothesis had no empirical support. A reasonable conclusion to draw is that both social causation and social drift are at play here, the former in the causation of 'schizophrenia' and the latter in its maintenance.

Poor people don't face more stress, they just can't deal with it

Some psychiatrists have argued that it is not the disadvantages accompanying poverty that are responsible, and have claimed instead that poor people are oversensitive to stress and therefore can't deal with it. Some have even argued that poverty doesn't involve exposure to disproportionate amounts of stressors. For example, researchers who found ten-fold differences in 'schizophrenia' between the poorest and wealthiest parts of Nottingham saw 'no clues' about causes: 'There was no particular suggestion of unusually high rates of stressful events' (Giggs & Cooper, 1987, p. 633). Others have claimed 'there is little evidence that lower-status individuals suffer from more situationally induced stress' (Rushing & Ortega, 1979, p. 1192).

It has even been argued that 'at any given level of stress, people of lower social class position are more likely to become mentally disturbed than are people of higher social class position'.

From this it is concluded that the 'relationship of class to schizophrenia is not attributable to the amount of stress that people endure. There must also be important class differences in how effectively people deal with stress.' In particular the poor have inadequate 'conceptions of social reality' characterised by 'fearfulness and distress, and by a fatalistic belief that he is at the mercy of forces beyond his control and often beyond his understanding' (Kohn, 1976, p. 179). Paranoia, it seems, is sometimes a heightened state of awareness. If poor people do have limited ways to deal with stress, this is, to a large extent, just another consequence of being poor.

In a rather extreme example of the power of dominant paradigms to influence its adherents to act in ways that protect the paradigm from incongruent realities, it was seriously proposed that no further research be undertaken on the topic, and that researchers should 'look for class-constant stresses, not stressors that are more frequent in the lower class, such as events related to the economy' (Eaton 1980). It seems this blatantly ideological recommendation has not gone entirely unheeded. Other reviewers concur with our earlier comments in concluding that 'Societal influences have rarely been addressed in recent reviews of schizophrenia' (Bresnahan & Susser 2003, p. 8). These particular reviewers, however, argue that 'socioeconomic status has at most a modest effect on risk of schizophrenia' and that 'no clear findings have emerged' (p.5). Other reviewers concur with the current author's reading of the research, i.e. that both social causation and social selection processes are clearly operating (Mohler & Earls, 2002).

Biased diagnoses

There is another reason, besides social causation, why poor people have a higher chance of ending up diagnosed 'schizophrenic'. By 1977 there were nine studies showing that more severe diagnoses are applied to poorer people than wealthier people *with the same symptoms* (Abramowitz & Dokecki, 1977). A tenth study found that psychiatrists assigning severe diagnoses on the basis of class genuinely believed they were basing diagnoses on 'patient'

behavior' rather than 'occupation and education'. Thus there was 'intellectual denial of social status effects and a subconscious utilization of status information' (Lebedun & Collins, 1976, p. 206). British clinical psychologist Lucy Johnstone concluded:

A number of studies have found that severer diagnoses are given to working- than to middle-class patients, regardless of symptoms; that the former are seen as having a poorer prognosis; and that professionals are less interested in treating them. (Johnstone, 2000, p. 238)

Public Opinion

The idea that poverty, and all its attendant adversities, can cause 'schizophrenia', while still contentious for some mental health experts, is not controversial to the public. In 16 countries where surveys about the causes of 'schizophrenia' have been conducted, the public (including patients and their family members) place more emphasis on psycho-social causes than bio-genetic factors (Angermeyer & Matschinger, 2005; Magliano et al., 2009; Read, et al., 2006). For example, the most endorsed causal model of 'schizophrenia' amongst Londoners was 'Unusual or traumatic experiences or the failure to negotiate some critical stage of emotional development', followed by 'Social, economic, and family pressures.' (Fumham & Bower, 1992). In a survey of over 2,000 Australians the most frequently cited cause (94%) was 'Day-to-day-problems such as stress, family arguments, difficulties at work or financial difficulties' (Jorm et al., 1997).

Campaigns (often funded by drug companies) to persuade the public to drop their belief that bad things happening can drive you mad and accept instead that 'schizophrenia' is a biologically based 'illness', have been shown to increase fear and prejudice rather than, as claimed, reduce it (Dietrich et al., 2004; Read, 2007).

Treatment

Class bias operates not only in the

diagnostic process but in treatment selection as well. Hollingshead and Redlich (1958) had found that 57% of class V (unskilled, manual) 'schizophrenics', but 24% of class I and II (business, professional and managerial) 'schizophrenics', received nothing more than 'custodial care'. Class V patients were more likely to receive physical treatments such as E.C.T, drugs and lobotomy. In the same population, ten years later, the poor were more likely to be hospitalised, were kept in hospital longer and received fewer treatments associated with discharge (Myers & Bean, 1968). A 1977 review confirmed that working class people were more likely to be treated with drugs than with psychotherapy (Abramowitz & Docecki, 1977). More recently, it has been concluded that:

Working class patients are, like black and ethnic minority patients, more likely to be prescribed physical treatments such as drugs and ECT, to spend longer periods in hospital regardless of diagnosis, and to be readmitted, and, correspondingly less likely to be referred for the more 'attractive' treatments such as psychotherapy or group therapy. (Johnstone, 2000, p.238)

For example, a recent survey of nearly 900,000 American children and adolescents showed that youths whose families are eligible for Medicaid (i.e. from poor families) are 5.8 times more likely to be on anti-psychotic drugs than those from families with private health insurance and, therefore, access to a broader range of treatments (Zito et al., 2003). A study of short-term inpatients in Alabama found that while race and gender did not predict discharge placement, more patients of lower socioeconomic class were transferred into long-term hospitalization (L. Hamilton, 2007). Adults from the more deprived areas of South Auckland, New Zealand (after adjusting for other demographic factors, diagnosis, chronicity, severity, psychiatrist and involuntary admission) had longer stays in psychiatric hospital than those from less deprived areas (Abas, Vanderpyl, Robinson, Le Proux, & Crampton, 2006).

Poverty is also a powerful predictor of compulsory admissions (Bindman, Tighe, Thornicroft, & Leese, 2002). For example, a study of the Auckland region of New Zealand found that compulsory admissions were highly correlated with neighbourhood levels of deprivation ($p < .0001$), and – again – that the strongest correlation was in the most deprived area, South Auckland (Kydd, et al., 1991).

Therapeutic and Policy Implications

In an earlier review of the literature, the current author put forward the argument that:

There appears to be a circle of oppression operating, in which biological psychiatry plays a crucial role. Of course the poor in any society are subjected to more sources of stress than the wealthy. In many societies poverty extends to hunger and homelessness. Even those with enough to eat and somewhere to live are more likely to experience powerlessness, isolation, lack of self-respect, physical ill-health etc. . . . Having entered the system they are more likely, regardless of their behaviour, to be hospitalised and labelled 'schizophrenic'. This is likely to further lower their self-esteem and motivation, and to frighten and distance loved ones. . . . They are less likely to be able to understand the real origins of their distress since this has all been explained away by their being 'schizophrenic'. They will, as a result, be even more powerless to change the circumstances that caused them to enter the psychiatric system in the first place. (Read, 2004e, p. 168)

It would, of course, be simplistic to lay all the blame at the door of biologically-oriented psychiatrists and the pharmaceutical industry that bankrolls their narrow paradigm. Some psychologists, including clinical psychologists, adopt an equally individualised, contextless approach to

research and clinical practice. Indeed clinical psychology largely abandoned the whole field of psychosis for forty years after the introduction, in the 1950s, of the first anti-psychotic drugs, preferring to focus on areas like depression and anxiety. Some of the leading cognitive psychologists, mostly British, who broke psychology's silence about madness in the 1990s, have paid proper attention to trauma (Bentall, 2003, 2009; Larkin & Morrison, 2006). Many psychologists, however, either continue to stay well clear of hallucinations and delusions or conceptualise such experiences as evidence of individual psychopathology rather than as understandable reactions to adverse life events. For example, there is an absence or paucity of family therapists in many mental health services throughout the world. In the psychosis field this may be partly explained by psychologists' fear of being accused of 'family blaming', a derogatory term frequently deployed by biological psychiatry and the drug industry for researchers and clinicians who adopt a family systems paradigm (Read & Bentall, in press; Read, Seymour, & Mosher, 2004). The continuing adherence of some psychologists to the idea that a diagnosis somehow explains the causes of a problem (e.g. the hallucinations are caused by the schizophrenia) may inhibit them from looking further or deeper for an explanation.

All mental health professionals will, if they ask the right questions (Read, Hammersley, & Rudegeair, 2007), and are not seduced by the delusion that counting symptoms and applying labels helps you understand what is going on for the person in front of you, repeatedly hear of the social causes of their clients' difficulties. Most of those social causes are related to poverty. Of course there are restraints on what an individual professional can do to help alleviate the poverty of an individual client or family, especially when the prevailing paradigm says its irrelevant and your agency tells you 'its not your job.'

We could, however, try a little harder. The chief of Public Psychiatry at the University of Chicago (19, p.1046) reminds us, that 'the seriously mentally ill are poor' and that:

With poverty and unemployment come all the other social consequences of being underclass – vagrancy, panhandling, substance abuse, and crime. Yet, with appropriate resources for supported housing and job placement, the panhandling, dishevelled, homeless mentally ill person can become just another neighbour. (Luchins, 2004)

We could certainly act on the growing body of research showing that psychological therapies, especially those with a greater focus on the therapeutic relationship and on the client's understanding of the origin and meaning of their difficulties (Bentall, 2009; Geekie & Read, 2009) are at least as effective as anti-psychotic drugs (Alanen, de Chavez, Silver, & Martindale, 2009; Bola, Lehtinen, Cullberg, & Ciompi, 2009; Morrison, 2009; Randal et al., 2009, 2010; Read, Mosher, et al., 2004a) and do not cause neurodegeneration, sexual dysfunction, obesity and diabetes, and do not increase cardiovascular risk or reduce life span (Bentall, 2009; Bentall & Morrison, 2002; Ross & Read, 2004; Weinmann & Aderhold, 2010; Weinmann, et al., 2009).

We could also use what the research and our clients tell us to try a little harder to lobby government to act in ways to reduce absolute and relative poverty, so as to help prevent 'schizophrenia' and other mental health problems in the next generation. It has been repeatedly argued that our efforts in this regard, and in establishing primary prevention programmes aimed at reducing poverty, should be targeted at the early years of life (Davies, Hanna, & Crothers, 2010; Perry, 1999). The brain differences between 'normal' and 'schizophrenic' adults, for decades cited as evidence that 'schizophrenia' is a brain disease which has little to do with childhood events, are found in the brains of children traumatised in the first few years of life (Braehler et al., 2005; Read, et al., 2001).

A common response to findings of high admission rates from deprived neighbourhoods, is to call for more inpatient beds in those areas (Abas, et al., 2003). Meanwhile 'prevention' in

the field of psychosis currently tends to mean identifying troubled teenagers who meet criteria for the recently invented notion of 'prodromal' psychosis and treating them as soon as possible, predominantly with anti-psychotic medication (Bentall & Morrison, 2002; Boyle, 2004). The huge recent increases in the prescribing of these drugs to adolescents that have resulted (Ross & Read, 2004) seem set to soar further if the proposal to introduce a new diagnosis called 'psychosis-risk syndrome' in the DSM-V is implemented (Morrison, Byrne, & Bentall, 2010; Ross, 2010). Other commentators, however, argue that 'this is the right direction to move in if we want to regain the space for a more psychosocial-based psychiatry' (Johannessen & McGorry, 2010)

If we were to take an evidence-based approach to the question of 'what should be done about schizophrenia?' we would be likely to conclude that the same primary prevention programmes to reduce child poverty, child abuse and neglect, and so on, that seem to have some effect on other health,

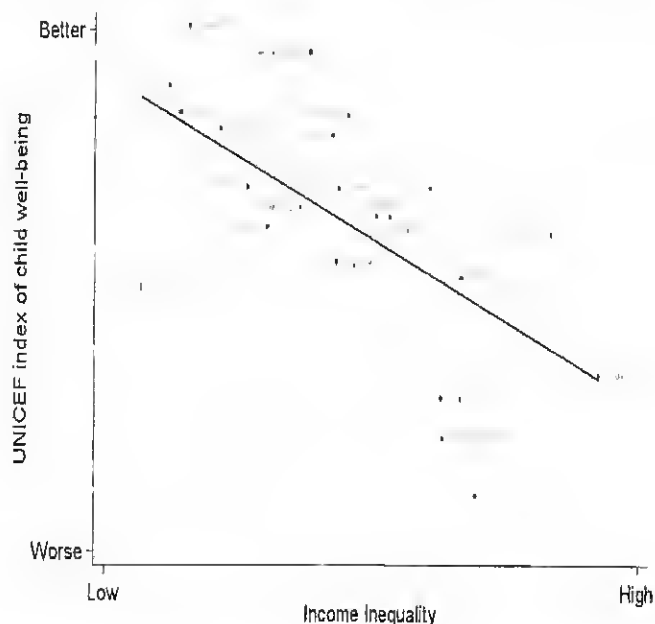
social and mental health outcomes, will work also to reduce the prevalence of madness. Persuading government to invest in the first five years of life will be just as important in relation to reducing psychosis as it is for other adverse outcomes. For example, an environmental enrichment programme at age 3-5 years has been shown to reduce schizotypal traits in early adulthood (Raine, Mellingen, Liu, Venables, & Mednick, 2003).

The case for primary prevention, and for a special focus on the first five years of life, has been repeatedly made, for years, all over the world, in relation to a broad array of outcomes. The only thing new, therefore, in this paper, is the research showing that, psychosis, 'schizophrenia' madness or whatever you prefer to call it, is, like most other mental health problems, largely caused by those social factors that are particularly common amongst the poor.

The researchers in the Dunedin Multidisciplinary Health and Development Research Unit, cited

Figure 3

Child Well-being is Better in More Equal Rich Countries



Source: Wilkinson & Pickett, *The Spirit Level* (2009)

Equality Trust

earlier, concluded:

Protecting children against the effects of socioeconomic adversity could reduce the burden of disease experienced by adults. These findings provide strong impetus for policy makers, practitioners, and researchers to direct energy and resources towards childhood as a way of improving population health. (Poulton, et al., 2002, p. 164)

The focus on *relative* poverty should not be forgotten when planning primary prevention programmes. Figure 3 shows the relationship between inequality and the 'Index of child wellbeing in rich countries', a measure combining 40 indicators compiled by UNICEF. The authors of 'The Spirit Level' conclude:

The solution to problems caused by inequality is not mass psychotherapy aimed at making everyone less vulnerable. The best way of responding to the harm done by high levels of inequality would be to reduce the inequality itself. Rather than requiring anti-anxiety drugs in the water supply or mass psychotherapy, what is most exciting about the picture we present is that it shows that reducing inequality would increase the wellbeing and quality of life for all of us (Wilkinson & Pickett, 2009, p. 33)

George Albee put it this way:

Psychologists must join with persons who reject racism, sexism, colonialism, and exploitation and must find ways to redistribute social power and to increase social justice. Primary prevention research inevitably will make clear the relationship between social pathology and psychopathology and then will work to change social and political structures in the interests of social justice. It is as simple and as difficult as that! (Albee, 1996, p. 1131)

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Sections of this paper are based directly on corresponding sections of a previous review by the same author (Read, 2004e), which the current paper updates.

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